



EDITORIAL

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Particulate Air Pollution and Mortality — Clearing the Air

In the years after World War II, several episodes of severe air pollution in the United States and Britain aroused public concern about the effects on health of air pollutants produced by burning fossil fuels. The most dramatic, the London fog of December 1952, caused thousands of deaths. Responding to concern about air quality in the United States, Congress passed the Clean Air Act in 1970. This act directed the Environmental Protection Agency (EPA) to identify pollutants that "may reasonably be anticipated to endanger public health and welfare" and to issue criteria for air quality that "allowing an adequate margin of safety, are requisite to protect the public health." Thus authorized, the EPA set National Ambient Air Quality Standards for particulate matter in 1971 and modified the standards in 1987. The 1987 standard was based on concentrations of particles with a mean aerodynamic diameter less than or equal to 10 μm , or PM_{10} . The maximum allowable 24-hour concentration was set at 150 μg per cubic meter, and the maximal allowable annual mean was set at 50 μg per cubic meter.

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In the United States, concentrations of particulate air pollution have declined since the early 1970s. From 1988 to 1993, the average of the annual mean PM_{10} concentrations at 799 sites monitored by the EPA declined by 20 percent.¹ Despite these improvements in air quality, a series of studies²⁻⁴ reported associations between particle concentrations and the numbers of deaths per day in several U.S. cities with mean 24-hour PM_{10} concentrations well below the standard. Responding to a substantial body of epidemiologic evidence, the EPA wrote in 1996 that the "staff can not conclude that the current standards protect public health with an adequate margin of safety" and that "fine fraction particles [$\text{PM}_{2.5}$, or particles with a diameter of less than 2.5 μm] are a better surrogate for those particle components linked to mortality and morbidity effects at levels below the current standards."¹ In 1997, the EPA retained the PM_{10} standards and promulgated new 24-hour and annual standards for $\text{PM}_{2.5}$ of 65 and 15 μg per cubic meter, respectively, based on consistency with the literature on health effects.

Both the epidemiologic evidence and the new $\text{PM}_{2.5}$ standard have been criticized. Some observers have asserted that the associations found in the epidemiologic studies are weak, inconsistent, and attributable to confounding by weather, other pollutants, or misclassification of the exposure to particulate matter.⁵ They note that many of the studies were performed by the same small group of investigators, that the study cities were not selected systematically, and that the statistical models varied from study to study.

Arguing that the 1997 standards for ozone and particulate matter did not have an adequate scientific basis, industry groups sued the EPA in the Court of Appeals for the District of Columbia. In 1999, the court blocked implementation of the 1997 standards. In its current session, the Supreme Court is hearing an appeal of this decision by the EPA.

A key issue before the Court is whether the cost of compliance can be considered in setting the standard. The EPA has estimated that compliance with the 1997 standards for $\text{PM}_{2.5}$ and ozone will require an investment of about \$10 billion per year to modify power plants, diesel trucks, and other sources of these pollutants⁶ and will result in health benefits with an estimated value of \$20 billion to \$100 billion per year. Others have estimated that the costs of compliance could be as high as \$60 billion per year.⁷ Thus, the Supreme Court's decision could have substantial consequences for the economy and the public's health. Given these stakes, the public and the scientific community need more and better information about the health effects of particulate air pollution.

The study reported by Samet et al.⁸ in this issue of the *Journal* (along with the more extensive investigation from which it is derived⁹) strengthens our understanding of the epidemiologic evidence and addresses the criticism of earlier work. The investigators used a single analytic approach to examine the association between PM_{10} concentrations in a given 24-hour period and the numbers of deaths reported on the following day in 20 of the largest cities and metropolitan areas in the United States. Samet et al. found an average increase in the rate of death from all causes of about 0.5 percent for every increase in the PM_{10} concentration of 10 μg per cubic meter.

The PM_{10} concentrations were positively associated with daily mortality rates in most of the 20 cities studied and at concentrations well below the current 24-hour standard of 150 μg per cubic meter. In fact, the 90th percentile of the distribution of daily values was below the 24-hour standard in each of the 20 cities. Moreover, the association was specific to PM_{10} . The concentrations of other regulated air pollutants produced by the combustion of fossil fuels (sulfur dioxide, nitrogen dioxide, and carbon monoxide) were weakly and inconsistently associated with daily mortality rates. Though ozone concentrations were positively associated with daily mortality rates during the summer months, this relation did not influence the association between the PM_{10} concentration and the daily mortality rate. Finally, the finding of a strong association between the PM_{10} concentration and the rate of death from cardiovascular and respiratory

causes offers support for the idea that the concentrations of particulate air pollution influence mortality.

The findings of Samet et al. are consistent with those of time-series studies in Europe¹⁰ and cohort studies in the United States.¹¹ Thus, the evidence in support of an association between the concentration of particulate air pollution and the mortality rate is consistent, is not affected by differences in statistical methods, and can be generalized.

There are important gaps in both the scientific evidence of causation and the scientific basis for the regulatory response. The most important is our inability to explain how fine particles affect health. Some studies have found that the daily mortality rate is associated with the concentration of fine particles ($PM_{2.5}$) but not coarse mass ($PM_{10}-PM_{2.5}$).¹² These findings are consistent with the evidence that fine particles penetrate indoor spaces, are chemically active, and are deposited in the respiratory bronchioles and alveoli. Yet little is known about the specific constituents or characteristics of $PM_{2.5}$ that adversely affect health. Moreover, although the standard proposed in 1997 is based on concentrations of $PM_{2.5}$, most of the epidemiologic evidence has been obtained from measurements of PM_{10} or other, less relevant indicators. Lacking knowledge of the harmful constituents of fine particles and the mechanisms by which they affect health, the EPA continues to propose standards based on particle mass.

The epidemiologic evidence suggests that the association between fine-particle concentrations and mortality is linear across the entire range of current concentrations. Although substantial reductions can be achieved at a reasonable cost, a reduction in 24-hour exposures to levels consistently below the current range would be prohibitively costly, if not impossible, in the foreseeable future. An aggressive research program to identify the harmful components of $PM_{2.5}$, their sources, and the mechanisms of their effects offers the best hope for developing more focused regulatory strategies that will simultaneously protect the public health and the nation's prosperity. In the meantime, these results present a challenge to policy makers who are required to protect the public's health with an adequate margin of safety.

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